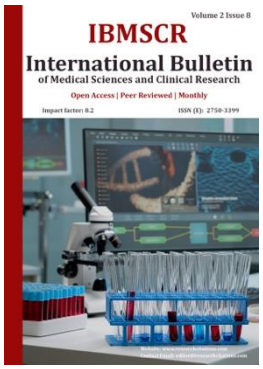

ACUTE CORONARY SINDROM AND RAPID EARLY ACTION FOR CORONARY TREATMEN

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Abstract: Lack of timely contact with the health care system leading to out-of-hospital death or delayed presentation remains a major obstacle to more effective management of patients with AMI.

Key words: reperfusion, arrhythmias, mortality, troponin, STEMI, NSTE-ACS.

Clinical trials of therapy for acute myocardial infarction (AMI) demonstrate the potential to limit infarct size and decrease mortality by coronary reperfusion and arrhythmia control. The effectiveness of these treatments is dependent on time and access to acute medical care. Coronary reperfusion requires early administration of thrombolytic agents or angioplasty.Defibrillation and other methods to control cardiac arrhythmias require trained personnel and equipment in an appropriate clinical setting. Although the need for timely receipt of these therapeutic strategies is well recognized, rapid access to emergency medical care remains a major problem. The majority of time lost is the period from the onset of symptoms to presentation in a medical facility, sometimes referred to as patient delay. Even when the decision is made to seek medical care, most patients in the United States avoid ambulance services, preferring self-transport. These delays, which average several hours, prevent the early application of life-saving procedures and contribute substantially to a diminished effectiveness of treatment.

Lack of timely contact with the health care system leading to out-of-hospital death or delayed presentation remains a major obstacle to more effective management of patients with AMI. The majority of CHD patients who die from cardiac arrest die outside the hospital without receiving medical attention. For those who experience AMI, delay in treatment is the major factor limiting the effectiveness of contemporary management approaches.Delay in seeking care is commonly divided into 3 major components: prehospital patient delay (time from symptom onset to seeking medical care); transportation delay; and delay in hospital evaluation and diagnosis prior to treatment.

Acute coronary syndrome (ACS) is associated with high mortality rates. Although the goal was to achieve a missed diagnosis rate of << 1%, the actual data showed a rate of >> 2%. Chest pain diagnosis has remained unchanged over the years and is based on medical interviews and electrocardiograms (ECG), with biomarkers playing complementary roles. We aimed to summarize the key points of medical interviews, ECG clinics, use of biomarkers, and clinical scores, identify problems, and provide directions for future research. Medical interviews should focus on the character and location of chest pain (is it accompanied by radiating pain?) and the duration, induction, and ameliorating factors. An ECG should be recorded within 10 minutes of the presentation. The serial performance of an ECG is recommended for emergency department (ED) evaluation of suspected ACS. Characteristic ECG traces, such as Wellens syndrome and De Winter T-waves, should be understood. Therefore, troponin levels

in all patients with suspected ischemic heart disease should be examined using a highly sensitive assay system. Depending on the ED facility, the patient should be risk stratified by serial measurements of cardiac troponin levels (re-testing at one hour would be preferred) to determine the appropriate time to perform an invasive strategy for a definitive diagnosis. The diagnostics should be based on Bayes' theorem; however, care should be taken to avoid the influence of heuristic bias.

In the last 10 years clinicians have been faced with the dilemma which platelet P2Y12 receptor antagonist to choose for the treatment of patients with acute coronary syndrome (ACS). This is because two P2Y12 receptor antagonists, prasugrel1 and ticagrelor2 , had shown superior efficacy over their predecessor, clopidogrel, in large scale randomized clinical trials and received a class I recommendation in the guidelines.3 The decision making process was further complicated by the fact that, according to guidelines, the 2 new drugs were to be administered in 2 different ways in patients with non-ST-segment elevation ACS (NSTE-ACS). Available evidence supports routine initiation of ticagrelor as soon as the diagnosis of NSTE-ACS was established without waiting for the results of diagnostic angiography.2,3 The recommendation is different for prasugrel. In patients with NSTE-ACS, initiation of prasugrel was dependent on the findings of diagnostic angiography.1,3 The reason behind are the results of a specifically designed trial that showed that pretreatment with prasugrel was not only not beneficial but also harmful in patients with NSTE-ACS.4 The lack of head to head comparison trials of ticagrelor vs. prasugrel given over one year for the whole spectrum of ACS made difficult the decision of whether to embrace a ticagrelor-based strategy with routine pretreatment or a prasugrel-based strategy that requires waiting for the results of diagnostic angiography before starting prasugrel treatment in patients with NSTE-ACS. The Intracoronary Stenting and Antithrombotic Regimen: Rapid Early Action for Coronary Treatment (ISAR-REACT) 5 trial was an investigator-initiated, randomized, multicenter, open label trial that compared a ticagrelor- with a prasugrel-based strategy in 4,018 patients.5,6 The study hypothesis was that ticagrelor is superior to prasugrel. However, contrary to our expectation, the primary endpoint of death, myocardial infarction, or stroke at one year was observed significantly more frequently with the ticagrelor-based strategy (9.3% in the ticagrelor group 6.9% in the prasugrel group, hazard ratio, 1.36; 95% confidence interval [CI], 1.09 to 1.70; P = 0.006, Figure 1). The superiority of prasugrel was consistent across the ACS subsets irrespective of whether the patients presented with ST-segment elevation myocardial infarction (STEMI) or NSTE-ACS. Importantly, the increased efficacy of prasugrel (26% reduction in the risk of the primary endpoint) did not occur at the expense of an increased bleeding risk. Major bleeding (defined as Bleeding Academic Research Consortium (BARC) type 3-5) was observed in 5.4% of patients in the ticagrelor group and in 4.8% of patients in the prasugrel group (hazard ratio, 1.12; 95% CI, 0.83 to 1.51; P = 0.46; Figure 1).6 The ISAR-REACT 5 trial provides two main lessons: Firstly, prasugrel is a more effective antiplatelet drug than ticagrelor in ACS patients. The best clue to this is provided by the subset of STEMI patients in whom pretreatment was the starting strategy for both study drugs. Secondly, pretreatment with ticagrelor does not offer any advantage in patients with NSTE-ACS. Although there was no study arm without ticagrelor pretreatment in the ISAR-REACT 5 trial, taken together, our trial and the A Comparison of prasugrel at the time of percutaneous Coronary intervention Or as pre-treatment At the time of diagnosis in patients with non–ST-segment elevation myocardial infarction (ACCOAST) trial offer evidence that pretreatment is

not needed in this subset of ACS patients.A few major characteristics of the ISAR-REACT 5 trial should be taken into consideration before extrapolating its results to the everyday practice. Notably, the study population in this trial is characterized by a very high proportion of patients treated with PCI and a very low proportion of patients treated with CABG or conservatively. Having said that, the results of the ISAR-REACT 5 trial are going to simplify the antiplatelet treatment algorithm of patients with ACS, making of prasugrel – in an individualized dose regimen – the mainstay of this treatment in all cases without specific contraindications to its use. While the diagnosis of STEMI is relatively straightforward and allows for starting prasugrel at the time of admission, in all patients with NSTE-ACS, initiation of prasugrel is dependent on the findings of diagnostic angiography.

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